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CORONARY PLAQUE CHARACTERISTICS IN PATIENTS WITH HYPERTENSION: A 3-VESSEL OPTICAL COHERENCE TOMOGRAPHY STUDY

Moderated Poster Contributions
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Background: Hypertension (HTN) is a well established risk factor for coronary artery disease. However, its relationship with coronary plaque morphology has never been systematically investigated in vivo. The present study aimed to explore the associations between HTN and coronary plaque characteristics using 3-vessel optical coherence tomography (OCT) imaging.

Methods: A total of 261 patients with coronary artery disease who underwent 3-vessel OCT imaging were included. Patients were divided into: HTN group (n=171) and non-HTN group (n=89). Culprit and non-culprit plaque characteristics were compared between the two groups.

Results: A total of 789 plaques were analyzed. At the non-culprit lesions, the prevalence of lipid-rich plaques (LRP) and thin-cap fibroatheroma (TCFA) was not different between HTN and non-HTN groups (LRP: 54.0% vs. 59.0%, p=0.225; TCFA: 9.9% vs. 12.6%, p=0.296). Non-culprit plaques in the HTN group showed significantly higher prevalence of calcifications (54.6% vs. 44.3%, p=0.012). In addition, protruding calcifications (14.3% vs. 6.6%, p=0.001) and cholesterol crystals (17.5% vs. 9.0%, p=0.001) were also significantly more frequent in the HTN group compared to the non-HTN group. At the culprit lesions, the prevalence of TCFA was significantly lower in HTN group than in non-HTN group (19.0% vs. 37.1%, p=0.038). Calcifications were more frequently observed in HTN than in non-HTN group (60.8% vs. 40.0%, p=0.040). At multivariate analysis, HTN was an independent predictor for the presence of protruding calcification at the non-culprit lesions (OR, 2.53 [95% CI, 1.05–6.07]; p=0.038), and was inversely associated with TCFA at the culprit lesions (OR, 0.29 [95% CI 0.08–1.04, p=0.057].

Conclusion: Patients with HTN had coronary plaques with higher prevalence of calcifications, particularly with a protruding pattern. Although TCFA was similarly distributed in the non-culprit plaques of patients with and without HTN, its prevalence was significantly lower in the culprit plaques of patients with HTN.